Physiological and Toxicological Aspects of Smoke Produced during the Combustion of Polymeric Materials

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Normally one expects that flame contact is the major cause of injury and death during fires. Analysis of the factors involved in numerous fires has revealed that most deaths were not due to flame contact, but were a consequence of the production of carbon monoxide, nitrogen oxides, and other combustion products, such as aldehydes, low molecular weight alcohols, hydrogen cyanide, and other noxious species.

The major emphasis within the scope of this paper relates to the physiological and toxicological aspects of smoke produced during the combustion of materials. Special emphasis is directed toward laboratory procedures which have been developed to determine the qualitative and quantitative analysis of smoke, factors pertaining to smoke development, and to measure the response of laboratory animals exposed to smoke. The effects that fire retardants, incorporated into polymeric materials as a means of improving flammability characteristics, may have on smoke development, the mechanism of polymer degradation, and on the survival response of laboratory animals are also considered.

Introduction

General Background

Normally one expects that flame contact is the major cause of injury and death during fires. Perhaps the first event focusing attention to the hazards of fire from plastic materials was that of the Cleveland Clinic fire in 1929, in which x-ray films, composed of highly combustible nitrocellulose, caught fire and brought death to 125 persons. Analysis of the death pattern revealed that most of the deaths were not due to flame contact but were a consequence of the production of carbon monoxide and nitrogen oxides. Since then numerous other fires in this country and abroad (Table 1) have also led to deaths, not only due to the actual flames, but to the gaseous products evolved

from synthetic materials. Not too surprising, however, is the fact that research on the toxicological aspects of pyrolysis and combustion during fire exposure has lagged so far behind other aspects pertaining to the flammability characteristics of materials, that even a fair assessment of the toxic hazards cannot be adequately described at this time except in great generalities. The time has passed for the toxic consequences during combustion to be ignored or minimized.

The major emphasis within the scope of this paper relates to the physiological and toxicological aspects of smoke produced during the combustion of materials. Since few definitive studies have been reported in the open literature pertaining to smoke and its effect on humans during and after fire exposure, this report also includes appropriate references and results of recent studies carried out in the Flammability Research Center of the University of Utah.

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Table 1. Summary of major fires in which smoke and toxic gases were considered to be a predominant factor causing loss of life.

Year	Place	Deaths
1929	Cleveland Clinic	125
1942	Cocoanut Grove Night Club (Boston, Mass.)	492
1963	Nursing Home (Fitchville, Ohio)	63
1965	727 Aircraft Crash (Salt Lake City, Utah)	43
1970	Harmar House Nursing Home (Marietta, Ohio)	22
1972	Sunshine Mine Disaster	92
1973	Isle of Mann Resort	50

Laboratory procedures which have been developed to qualitate and quantify smoke produced during thermal decomposition processes are also detailed. Finally the effects of fire retardants, both reactive and inert, incorporated into synthetic materials to enhance their flammability characteristics, are reviewed.

Definition of Smoke

A dictionary definition (1) of smoke is "the volatilized products of the combustion of an organic compound, as coal, wood, etc., charged with fine particles of carbon or soot; less properly, fumes, steam, etc."

Gaskill (2) defined smoke as "the airborne products evolved when a material is decomposed by heat or burning (oxidation)." He further states that "smoke may contain gases, liquid, or solid particles, or any combination of these."

Hilado (3) defined smoke as "the gaseous products of burning organic materials in which small solid and liquid particles are also dispersed; smoke can also be defined as solid particles, such as carbon and ash, suspended in air." Hilado further stated "that the broader definition is the more appropriate because the nongaseous portion of smoke from some materials contain significant amounts of tarry or liquid droplets." Thus, little difference is noted among these three definitions of smoke.

Factors Affecting Life Support in Fires

The major factors affecting life support in confined space fires, listed in the order of greatest damages to human survival, are: (1) reduction of oxygen concentration accompanied by increase in the concentration of carbon monoxide; (2) development of extremely high temperatures; (3) presence of smoke; (4) direct consumption by the fire; (5) presence of noxious or toxic gases; (6) the development of fear. From a toxicological point of view, factors (1), (3), and (5) become important considerations, since factors (2) and (4) will cause immediate death, while factor (6) may or may not lead to death, depending upon whether a panic-stricken person makes a rash decision such as jumping out of a building rather than waiting for rescue.

Exposure of humans to the various combustible gases normally encountered in fires as well as to the particulate matter in smoke may bring about acute episodes of toxicity, ranging from minor irritant effects to death.

A series of disasters in recent years has focused considerable attention on the growing list of problems connected with the burning of polymeric materials, such as fibers, coatings, elastomers, foams, and reinforced plastics. The crash of the United Airlines Boeing 727 jetliner at the Salt Lake City Airport in November 1965 was one of the most dramatic incidents illustrating the dangers arising from intense heat, toxic fumes, and dense smoke. This tragedy, which took the lives of 43 persons of the 91 aboard, was one of the rare instances of what the Civil Aeronautics Board termed "survivable crash with no fatalities on impact.'" Yet the question remained: What was the contribution of the plastic materials inside the plane to the development of fumes and smoke? Previous attempts by industry to fire retard plastics produced improved resistance to flame contact; however, hazards due to smoke generation were, in general, not fully understood and recognized.

Disasters of the Salt Lake City type clearly spell out the need for the use of plastic materials that exhibit both adequate flame resistance as well as low-smoke generation. The necessity exists, therefore, for the development of synthetic materials with these properties and the more accurate evaluation of such materials to permit the prediction of their behavior during fire exposure.

A critical analysis of the hazards to life support in fires involving plastics has been carried out. The burning process takes place in several steps. In the first step, a destructive

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distillation of the plastic takes place, producing gases whose nature depends on the composition of the material, temperature, and the rate of heating.

Next, oxygen combines with free carbon to form carbon monoxide. At this time dense smoke is usually formed, presenting additional hazards by limiting egress from the fire area. When sufficient oxygen is present, it combines with the flammable gases produced as well as with carbon monoxide. If sufficient excess oxygen is present to combine with all of the combustible materials, the carbon monoxide present burns to form the relatively harmless carbon dioxide. Ordinarily, the products of complete combustion are less harmful than those of incomplete combustion.

In his role as Deputy Fire Marshal for the State of Utah and Special Consultant to the Salt Lake City Fire Department, the author has served as an investigator for a number of fires in which severe injury or loss of life was attributed to smoke as well as toxic gases resulting from the combustion of materials commonly encountered during such conflagrations.

The fire in the Lil-Haven Nursing Home (September 15, 1971) in Salt Lake City, Utah serves as an excellent example to illustrate conditions encountered in many confined space fires. Six patients died in this fire, that lasted approximately 10 min. The first fire equipment arrived on the scene less than 1 min after the rate-of-rise detector signaled the alarm to the central fire station. It is interesting to note that none of the six victims who died showed any evidence of body burns. External views of the Lil-Haven Nursing Home taken after the fire show, with the exception of a slight trace of smoke damage in the eaves just under the roof, no outward evidence of a fire having taken place. The hollow-core wood panel doors found in the second-floor hallway of the nursing home were severely damaged on the exterior but were still standing, and did provide a suitable fire barrier. Firefighters found all of of the doors leading to the hallway in rooms located on the second floor open at the time of the fire.

Smoke and soot patterns were indicative of a fast-spreading fire in the interior of two of the rooms in which four of the victims died. The smoke patterns seem to corroborate the view that had these doors been shut, sufficient

protection would have been provided until this fire was brought under control so as to preclude the loss of life.

The propensity of certain materials to ignite and burn with a rapid propagation rate has encouraged industry producing these materials, as well as government agencies, to find ways of preventing or diminishing the ignition and flame-spread characteristics of these materials. Generally, polymeric materials contain chemical compounds, called fire retardants, to reduce the original flammability characteristics of the material. The use of fire retardants is increasing at a prodigious rate each year. Little relative evaluation is being carried out to ensure that these compounds are not contributing to the development of limiting toxicants during fire exposure. Unfortunately, as may happen on occasion, the treatment may bring about another hazard equal or greater to the problem which originally required the treatment. In this case, the flame retardants do improve the flammability characteristics of the materials, but by doing so, they increase the concentration and types of pyrolysis products which may be liberated during fire exposure. These degradation products, singly and synergistically, may have biological implications not previously appreciated.

Physiological Factors Affecting Survival during Fire Exposure

The factors that critically limit survival response during fire exposure must be defined. In actual fires it is difficult, if not impossible, to separate the physiological parameters from the toxicological parameters. Within the scope of this paper this separation has been made in order to elucidate that which is presently understood about the response of humans in fires and to indicate those areas where further study is required.

In many respects, it is more important to determine the limits for survival than the mechanism of death from exposure to noxious gases or hypoxia. Additional studies are required to determine the long-range effects of acute exposure to carbon monoxide, temperature, and a variety of interacting noxious and toxic by-products of combustion.

Visual Aspects Affecting Survival

Considerable attention has been directed to the measurement, by optical techniques, of the quantity of smoke produced during the combustion process. It should be noted that while many chemical and physical factors can affect the quantity of smoke produced, the size, and concentrations of particulates in the smoke, and the chemical composition of the smoke, the optical techniques commonly used can measure only the light-obscuring potential of smoke and cannot measure the physiological factors relating to human survival during fire exposure.

The particulates contained in smoke can and do affect the vision of firefighters using gas masks and sustained breathing equipment. The results obtained with the use of the Rohm and Haas XP-2 chamber (4) or the National Bureau of Standards smoke density chamber (5–8) can relate directly to the ability of firefighters to see under fire conditions. On the other hand, persons encountering smoke in a fire may not be able to see due to such factors as lacrimation caused by components in the smoke. Einhorn et al. (9) reported that certain fluorine-containing polymers exposed to pyrolysis or combustion caused severe opacification of the cornea in test animals exposed to their degradation products. The hydrogen fluoride, fluorine gas, and carbonyl difluoride, identified in the smoke, was in sufficient concentration to etch glass coverslips placed in the test chambers. Thus, even though relatively low concentrations of smoke were generated during the pyrolysis or combustion process, the sight of humans exposed in a similar environment may be impaired to the extent that egress from a fire in time to prevent exposure to lethal concentrations of toxic fumes or temperatures sufficient to cause death would be impossible.

The Escape Response

Reliable data concerning the escape response of humans during fire exposure are scanty. The influence of hypoxia alone and in combination with carbon monoxide and noxious gases must be determined both at ambient temperatures and at rates of temperature increase that are encountered in "typical" fires. Numerous investigators have reported cases where a victim has died during a fire with no visible barrier to prevent easy escape. Victims of fires, such as those caused by cigarette ignition of mattresses or furniture, where long periods of smoldering have preceded actual ignition, are often found away from the ignition source, as for example, a woman found near a doorway after causing a fire on a mattress by smoking in bed. It should be noted that chemical analysis of the blood showed a 0.265 mg-% blood alcohol level accompanied by 33% carboxyhemoglobin saturation. The soot pattern in this case showed the body outline on the floor, with no observable soot formation under the body, thus clearly indicating that death occurred prior to the onset of flaming combustion. The chemical analysis confirms that the combination of alcohol and carbon monoxide saturation, as well as increased temperature, may have been the cause of this death. But one must consider possible loss of sight prior to death, the possible effect of combustion products on nerve conduction velocity reduction, the effect of degradation products on muscle activation and contraction as factors possibly preventing escape from a hazardous area even though the victim realized that he must leave the fire area in order to survive.

Noxious gases which find their way into the circulation, either because they are odorless or are present in low concentrations, may act in still unknown ways in producing neuromuscular dysfunction. The peculiar affinity of carbon monoxide for hemoglobin and cyanide radicals for cytochrome oxidase are two well-known examples of this phenomenon. Other degradation products may also affect oxidative metabolism at various levels, either by influencing oxygen transport or intermediary metabolism. The enzyme systems concerned directly with muscle activation and contraction may also be affected.

Studies comparing responses to hypoxia or various types of CO interaction have disclosed significant differences. Since arterial P_{O_2} may be normal despite a reduced oxygen-carrying capacity, reflexes which normally increase respiratory rate and tissue blood flow may not be activated (10,11). Recovery from hypoxia associated with CO intoxication is greatly prolonged in comparison to recovery from hypoxia alone (12,13).

A reduced partial pressure of oxygen is found in the poorly ventilated environment in

which combustion occurs. Combined with the pressure of carbon monoxide and other gases impairing oxidative metabolism, very little reduction in ambient oxygen may be lethal. From observations of subjects at high altitude, lassitude and lack of motivation progressing to somnolence are primary behavioral responses during hypoxia (14). These effects threaten survival both at high altitude and during accidental fire. The ability to continue automatic motor activities, such as running, depends both upon the central nervous control of this activity and on the neuromuscular system. The ability of these systems to continue normal function during fire exposure must be determined. It should be possible to assess the relative importance of the central nervous and peripheral neuromuscular systems which may be responsible for loss of motor control.

Toxicological Aspects of Combustion

General Background

The most controversial and most complicated aspect of fire research is that phase of study directed toward an understanding of the toxicological properties of materials during fire exposure. Hundreds of articles have appeared in the literature reporting the nature and quantitative analysis of pyrolytic decomposition products. Bulletin 53 published by Underwriters' Laboratories (15) cites 297 references dealing with toxicity studies on animals exposed to a wide variety of environments under many experimental conditions. In the summary of this report it was stated that considerable variance was observed in the experimental results and that little correlation was obtained by different investigators.

If laboratory animals are subjected directly to the degradation products of materials at temperatures normally encountered in real fires, they may perish from the effects of heat before being overcome by the decomposition products.

In recent investigations, Einhorn et al. (16) studied the effect of temperature on lethality (LD.₀₀) of laboratory rats of varying body weights. A preliminary heat transfer mechanism was postulated. Although an induction period was observed due to the insulating characteristics of the animal's fur, the relatively

large surface-to-volume ratio resulted in their inability to survive extensive thermal shock to the degree that humans can.

A series of studies are being conducted under a National Science Foundation-RANN Grant. These experiments have been designed to determine the physiological effects of smoke generated during the pyrolysis and combustion of polymeric materials on laboratory animals. Initial experiments were designed to determine the maximum levels for temperature exposure before permanent changes are induced in vital functions. A maximum body temperature of 42°C for extremely brief durations has been established as a ceiling for Sprague-Dawley rats. Exposures at this level will produce major dysfunction in the central nervous system, the respiratory system, and possibly other systems.

Previous studies by many investigators have suffered from several defects in design. First, the number of animals was small. Second, there was often a lack of control. Third, there were few standardized pre- and post-exposure tests of the animals' responses. Fourth, routine necropsy examinations of all major organs were not the rule.

A further weakness in many investigations pertaining to the toxicological aspects of combustion is the failure to simulate conditions of common prototype fires such as aviation fires, home fires, automobile fires, and boat fires, etc. For example, the probable time of exposure and the conditions of exposure, such as temperature and oxygen concentration, have not been key factors considered in experimental design.

Analysis of decomposition products resulting from pyrolysis or combustion has indicated major changes in the nature of the products due to condensation, recombination, or cross-reactions where the temperature of combustion is modified. Experimental design considerations must also be given to changes in the degradation of a single material as compared to the degradation of the same material in the presence of one or more materials of different chemical composition.

Respiratory Burns

In 1962, Phillips and Cope (17) labeled respiratory tract damage as "a principal killer" in burn victims. In 1967, Stone and Martin (18) reported respiratory involvement in 15%

Table 2. Burn mortality: New York City, 1966 and 1967.

Post-burn survival time, hr	Total cases by survival time		Autopsied cases (311)	
	Cases	%	Cases	%
<12	283	53	185	60
>12	158	30	72	23
Not known	93	17	54	17
Totals	534	100	311	100

of 197 burn patients studied. Zikria (19) analyzed the causes of death among fire fatalities which occurred in New York City during the years 1966 and 1967. Table 2 presents a summary of this study. Analysis of the data in Table 2 shows that 311 of 534 fire victims were autopsied; of these, 60% died at the site of the fire or on the way to the hospital. Of these early fatalities, 70% had respiratory involvement.

As many as 105 of the fire fatalities had less than 40% body burns; 77% of these victims could have been expected to survive, if statistical predictions were based solely on the extent of body surface burns.* Respiratory involvement was found among the majority of these fatalities: specifically, smoke poisoning and/ or asphyxiation, 43%; carbon monoxide poisoning, 50%; damage to the tracheobronchial tree and lungs, 27%. These figures clearly indicate the magnitude and seriousness of the problem of inhalation injuries to fire victims.

It is generally accepted that tissues of the tracheobronchial tree and pulmonary tissues can sustain heat damage, chemical damage, anoxic damage, or any combination of these injuries during fire exposure. Pressure damage may also occur when the fire is accompanied by an explosion.

Until the late 1960's many investigators doubted that caloric inhalation damage could

* Medically the "rules of nine" (20) are used to express the extent of a burn. One arm is 9%, a leg is 9%, front and back 9%, etc. The percentage of the body involved is important for both treatment and plotting survival figures. If a third degree burn involves 50% of the body surfaces, the mortality rate is about 50%. If a third degree burn involves 70% or more of the body surfaces, survival is nil. A healthy adult may survive a 10–15 percent third degree burn without too much difficulty; a healthy child may survive a 5–10% third degree burn without too much difficulty.

occur in the tracheobronchial tree because of the low specific heat of gases. Moritz (21) conducted experiments on dogs using high-temperature torches as the source of combustion. This study seemed to indicate the physical impossibility of caloric damage. In 1968, Zikria et al. (22) and Stone (23) demonstrated heat fixation of the tracheal mucosa in fire victims as well as the presence of varying degrees of injury to the tracheobronchial tree. All of the victims who had severe tracheobronchial damage were dead at the scene of the fire or soon thereafter.

Smoke Poisoning

Zikria (19) indicated that smoke poisoning was a primary diagnosis in 119 victims of the 185 early burn fatalities studied (Table 3).

Lethal levels of carbon monoxide poisoning were discovered in 45 of the 185 early deaths studied by Zikria (22) (Table 4).

Table 3. Respiratory tract complications in 257 autopsied cases.

	Post-burn survival time <12 hr		Post-burn survival time >12 hr	
	Cases	%	Cases	%
Smoke poisoning and/ or asphyxia only	99	53.5	4	5.6
Respiratory tract damage and/or pulmonary damage only	11	5.9	28	38.9
Both	20	10.8	1	1.4
Neither	55	29.9	39	54.1
Total	185	100.0	72	100.0

Table 4. Carbon monoxide poisoning in 185 autopsied cases with death occurring in less than 12 hr.

	Carboxyhemoglobin saturation, %	No. of cases	%
Laboratory determination (total)		(130)	(70.3)
Usually lethal	>50%	45	24.3
Significant	11%–49%	64	34.6
No contribution	7%–10%	21	11.4
Clinical diagnosis only		14	7.6
No indication		41	22.1
Total		185	100.0

In real fire exposure it becomes nearly impossible to ascertain which one or two agents (excluding absence of oxygen, presence of carbon monoxide, and perhaps direct evidence of large quantities of particulate matter in the upper respiratory tract) caused death. It becomes even more difficult to ascertain the toxic potential of a specific material when it burns or is heated. Presently, the most widely used approach is to have some knowledge of what gases are formed and to seek toxic information (LD_{50}) on the individual compounds if such knowledge is available. This method of evaluating the potential hazard of a given species of toxic gas leaves much to be desired. Little useful information can be gained from a LD_{50} study; for example, this type of study measures only death, does not relate the effect the toxicant may have on the survival response, and does not consider the possible long-term effects which do not manifest themselves during the normal 14-day observation period. LD_{50} experiments shed little light on the mechanism of death and thus do not provide information necessary for a system or engineering design for fire safety.

The problem of identifying the role of a single product on life support or the survival response becomes greatly magnified, since the combination of products being inhaled may not, and generally do not, produce the same biological response as when only one of the compounds is inhaled.

When man is placed in contact with a chemical agent, it can produce an acute toxic effect in a number of ways: the compound may act as a primary irritant upon the skin and/or mucous membrane; the compound may be absorbed into the blood stream, leading to definite toxic symptoms and signs, and which may result in death on continued exposure; the compound may be absorbed in very low concentrations, producing no definite signs and symptoms of toxicity, but may affect mental functions; the compound may act as a sensitizing agent, producing antibodies to the antigen. A repeated exposure to the same, or nearly similar compound, may produce allergic manifestations ranging from mild to very serious.

The first two can lead to rapid death during fire exposure, or if not death, may result in sufficient damage to cause hospitalization. Low levels of a compound may be sufficient to alter mental functions, and this, in turn, may lead

to serious consequences. The fourth (sensitizing agent) consequence has not received much attention in regard to fires, but should not be overlooked, at least in those cases where death does not occur or even in those instances in which no harm is evident. The burning or the pyrolysis of a polymeric material may, however, initiate an allergic response which becomes manifest at a later date. Such injury is often reported; approximately 10% of those individuals exposed to carbon monoxide seemingly recover and some months after actual exposure, develop a major dysfunction which may lead to death.

The Fire Gas Research Report (24) evaluated the effect of oxygen concentration as it pertains to human response during fires. Table 5 summarizes the signs and symptoms of toxicity caused by reduced levels of oxygen due to fire conditions.

Shorter et al. (25) reported that temperatures in excess of 300°F (149°C) were capable of causing loss of consciousness or death within several minutes. The temperatures recorded in several controlled experimental fires in buildings exceeded the maximum survivable levels within 5–10 min. This period of time is expected to be greatly reduced, for instance in aircraft fires, due to the large concentrations of available fuel, and thus egress from the cockpit area must be achieved within approximately 90 sec if survival of the passengers is to be realized.

Smoke development measurements have been made by a number of experimental techniques. Dense smoke discharged into the atmosphere by burning wood, cotton, paper, or plastics contain many toxic and noxious products resulting from thermal decomposition including carbon monoxide, carbon dioxide, hydrogen cyanide, hydrogen halides, and a large number of organic irritants, such as acetic acid, formic

Table 5. Consequences of toxicity of reduced levels of oxygen due to fire conditions.

Oxygen in air, %	Signs and symptoms
>20	None
12–15	Muscular coordination for skilled movements lost
10–14	Consciousness continues; judgment is faulty, muscular effort leads to rapid fatigue
6–8	Collapse; reversible with prompt treatment
≤6	Death in 6–8 min

acid, formaldehyde, acetaldehyde, acrolein, low molecular weight ketones, and alcohols. During the early stages of a fire, the smoke may contain so little carbon monoxide that the major injuries resulting from smoke inhalation may be caused by these irritants. These attack the mucous membranes of the respiratory tract and create conditions favoring the onset of pneumonia. In cases of actual exposure, the physiological effects of inhaling smoke depend upon its physical state. When smoke is very hot, it will destroy the tissues by burning, regardless of its chemical composition; when cooled, the smoke may be nonirritating because the irritants may have been removed by condensation and settling. Consideration must also be given to the size of the particulates entrapped in the smoke. If these materials are large, they may be easily removed by ciliary action within the body. If, however, they are small, the removal process may become more difficult. Smoke studies conducted by personnel of the University of Utah's Flammability Research Center have shown that many of the particulates produced during the combustion of natural and synthetic materials have average particle size ranging from 0.1 to 0.5 μm. Another factor may play an important role in human response to smoke. Samples of particulates screened from smoke generated during pyrolysis or combustion of polymeric materials have been examined by using the techniques of electron spin resonance. All the evidence indicates that many of the free radicals, normally having extremely short half-lives, may be entrapped in the smoke particles, thus strong signals are obtained for periods as long as 5 or 6 weeks. Preliminary tissue culture experiments have shown the development of abnormal nuclei when subjected to particulates having entrapped free radicals. This work is in its very early stages, and considerable additional experiments must be conducted before any definite conclusions can be drawn.

In addition, dense smoke may prevent exit from the area in which a fire is located by obscuring vision. This same obscuration effect may prevent location of the source of the fire and thus hinder fire control.

Many investigators have conducted studies on single materials under controlled laboratory pyrolysis or combustion conditions. In actual fires, combustion of single materials is seldom encountered, and there is ample evidence to show that the sum of the toxicity potential of two or more gases or vapors may be synergistically enhanced. In a fire, the toxicity of such mixtures may be further increased by low oxygen concentrations and high temperatures. Carbon dioxide, for example, causes stimulation of the respiratory center of the brain, and if inhaled in excess during a fire, it causes an abnormally high intake of other toxic gases which may prove fatal.

Although the lungs and associated structures are principal sites of action for irritant fire gases, corrosive vapors such as acids and acetaldehyde will also affect the unprotected skin and the cornea of the eye. Whatever the tissue exposed, the effect will cause inflammation. If the concentration of irritant gas or vapor is high or the exposure prolonged, fluid drawn from the blood and tissues accumulates in the respiratory organs. This condition is called tracheal, bronchial, or pulmonary edema, depending on the level in the respiratory tract which is affected.

Toxic Effects of Gases and Thermal Degradation Products

Oxygen

The important factor is the absence of oxygen rather than the release of oxygen due to burning or pyrolysis of polymeric materials. Complete lack of oxygen will lead to death within a few minutes, and decreased concentrations of oxygen in the air will produce a number of signs and symptoms of hypoxia in persons exposed to that environment. Even if death does not occur due to the lowered levels of oxygen in the immediate atmosphere, insufficient supply of oxygen to brain tissues for short periods of time will produce irreparable brain damage. Concentrations of oxygen higher than this but still below normal ambient levels will affect the brain cells in a reversible manner, but during this period the person will have behavioral changes which may produce faulty judgment, the consequences of which may be grave injuries to himself as well as to others (Table 5).

Carbon Monoxide

Of all of the gases generated in burning of organic materials (both natural and synthetic),

Table 6. Signs and symptoms at various concentrations of carboxyhemoglobin.

CO-Hb,%	Signs and symptoms
0-10	None
10-20	Tightness across forehead, slight headache, dilation of the cutaneous blood vessels
20-30	Headache and throbbing in the temples
30-40	Severe headache, weakness, dizziness, dimness of vision, nausea, vomiting, collapse
40-50	Same as above, greater possibility of collapse; syncope, increased pulse and respiratory rates
50-60	Syncope, increased respiratory and pulse rates, coma, intermittent convulsions, Cheyne-Stokes respiration
60-70	Coma, intermittent convulsions, depressed heart action and respiratory rate, possible death
70-80	Weak pulse, slow respiration leading to death within hours
80-90	Death in less than 1 hr
>90	Death within minutes

Table 7. Physiological response to various concentrations of carbon monoxide.

	Concentration of CO in air (by volume)	
	ppm	%
Allowable for an exposure of several hours	100	0.01
Without appreciable effect if inhaled for 1 hr	400– 500	0.04–0.05
Just appreciable effects after 1 hr of exposure	600– 700	0.06–0.07
Unpleasant but not dangerous symptoms after 1 hr	1000–1200	0.10–0.12
Dangerous in exposure for 1 hr	1500–2000	0.15–0.2
Fatal in exposures of less than 1 hr	≥4000	≥0.4

importance as compared to combining with hemoglobin. Both animal and human studies have demonstrated that correlations can be made between signs and symptoms of toxicity and the percentage of CO-Hb formed. Table 6 (26) summarizes this information and shows that CO-Hb concentrations below 10% produce no signs or symptoms. In general, most people will not show toxic symptoms below a level of 20% carboxyhemoglobin. From this level on, however, extremely toxic manifestations will occur at CO-Hb concentrations of 60% or more, and death is imminent. Henderson et al. (27) reported on the physiological response to various concentrations of carbon monoxide (see Table 7).

In order to determine the relationship between the concentration of carbon monoxide in the air and the CO-Hb content, several investigators have exposed laboratory animals at various concentrations of carbon dioxide and measured the time required to reach a given blood level. Table 8 summarizes the work of Hofmann and Oettel (28) pertaining to the blood CO-Hb levels between rat and man.

It should be recognized (as illustrated in Table 8), that saturation curves for humans are not directly applicable to rats, because these animals inhale a larger volume of air per unit time in relation to their body weight, thus their blood can become more rapidly saturated with carbon monoxide. Considerable differences in the reported lethal concentrations of carbon monoxide are found in the literature. This may be due in part to the animals used, the conditions of exposure, or the methods used to monitor the environment.

the gas which has been thought to produce the most deaths in real fire situations is carbon monoxide. The air we breathe has levels of carbon monoxide in the parts per million range; animals and man apparently can tolerate concentrations up to 100 ppm for short periods of time (up to 8 hr) without undue harm. Fire conditions, however, can release large concentrations of CO into the air, and CO at these levels can lead to death in very short periods of time. The main action of carbon monoxide after it is inhaled is to combine reversibly with hemoglobin (Hb) to form carboxyhemoglobin (CO-Hb). This reaction displaces oxygen in the blood and leads to anoxia and death if the reaction is not reversed. Carbon monoxide also interferes with oxygen release in the tissues, but this appears to be of secondary

Table 8. Species differences with regard to CO-Hb concentration in blood.

CO concentration, ppm	Rat		Man	
	Time to 20% CO-Hb, min	Time to 50% CO-Hb, min	Time to 20% CO-Hb, min	Time to 50% CO-Hb, min
10,000				
5,000				
2,000		15	20	60
1,000	15	240	60	300
500	30	(•)	90	(•)
250	90	(•)	360	(•)

• 50% level not reached. Rats will die at 70% CO-Hb concentration within 30 min.

In recent years, attention has been given to possible toxic effects of carbon monoxide at levels where signs and symptoms of toxicity are not noted, as for example, below 10% CO-Hb. Schulte (26) has explored this problem and has found that concentrations as low as 5% CO-Hb can affect certain psychomotor abilities. For example, in experiments with humans he noted that both the rate of errors and the time needed to complete an arithmetical chore would increase. He also employed other tests in his human experiments and came to the conclusion that low levels of carbon monoxide could have, and most likely do have, an effect upon judgment and situational decisions and responses. Some investigators have reported that certain cigarette smokers may at times show up to 10% carboxyhemoglobin in their blood, depending upon the number of cigarettes smoked and the manner in which they are smoked. Other figures, however, generally show a level of less than 5%.

If low concentrations of carbon monoxide can indeed affect decision-making and other psychomotor responses, this may provide a possible answer for the inability of victims to escape an area if they have been exposed to high concentrations of carbon monoxide.

Carbon monoxide has 300 times the affinity for hemoglobin of oxygen. When carbon monoxide is included in inhaled air, the reaction (1) occurs in the lung cells:

$$O_2 \cdot Hb + CO \quad CO \cdot Hb + O_2 \quad (1)$$

Although this is a reversible reaction, as the affinity for carbon monoxide is stronger than for oxygen, oxygen hemoglobin ($O_2 \cdot Hb$) no longer can be formed. As the carboxyhemoglobin is formed, the hemoglobin loses its capacity to transport oxygen, resulting in an oxygen shortage in tissues and organs. It should be pointed out that there is strong belief by our medical personnel that a similar relationtion exists between carbon monoxide and myoglobin and thus, additional physiological effects may take place with regard to muscle activation and contraction, again limiting the ability of the victim to egress the fire area.

Consideration must also be given to methods which favor the rapid reversal of the concentration of carboxyhemoglobin in the blood. Einhorn et al. (9) exposed laboratory animals exhibiting convulsions during the agonal epi-sode, which is consistent with a cerebral hypoxia due to carbon monoxide poisoning, to pure oxygen. The convulsions ceased within 1 min and the animals were grooming themselves within 30 min. Also, Kishitani (29) exposed mice to fresh air after carbon monoxide exposure and observed a rapid return to normal carboxyhemoglobin levels. Packham (30) reported similar results using Sprague-Dawley and Long-Evans rats. He reported that the loss of avoidance response is not rigidly dependent upon a critical CO-Hb level, but rather is strongly affected by the rate at which the CO-Hb levels are reached and the protocol of the experiment.

In present studies the level of toxicity is described in terms important to survival of the animal, e.g., its ability to escape from a noxious environment. It is the object of this study to describe the physiological status of the animal at selected stages of toxicity.

Definition of Levels of Toxicity: For the purpose of the carbon monoxide experiments, five levels of toxicity have been defined by Petajan and Packham (31) as follows.

LEVEL 1, ATAXIA: Animal movements are *unsteady and inaccurate; titubation of the head and trunk occurs. Grooming behavior loses precision of movement.*

LEVEL 2, LOSS OF SURVIVAL RESPONSE: Rats conditioned to avoid a shock by bar pressing lose this response. The rat generally leaves the manipulandum and moves aimlessly about the chamber.

LEVEL 3, LOSS OF POSTURAL TONUS: There is progressive loss of postural tonus until the rat is flattened out upon the floor of the chamber. When this level of toxicity is reached, electric shock will produce local limb withdrawal, but neither movement to a different area of the chamber nor elevation of the body from the floor.

LEVEL 4, ANOXIC SHOCK: There is no response to electric shock other than local muscle contraction. Respiratory rate drops to approximately half the resting level either consistently or in brief epochs of bradypnea. Cardiac arrhythmia is seen at this stage.

LEVEL 5, DEATH.

LD_{50} versus Survival Response: The lethal dose—50 (LD_{50}) test protocol has been widely accepted by toxicologists for studying dose–response effects on lethality. This test protocol

has been widely used in the study of such things as drugs, pesticides, single gases, or mixtures of simple gases. This test has been designed to relate the concentration of the item being studied to an endpoint which involves the death of 50% of the test animals. In the dynamic environment of a fire exposure, the LD_{50} has many major shortcomings. For example, no indication is obtained to which gas or gases in a complex mixture of toxicants is the limiting agent. Further, counting the number of animals that succumb to a given exposure does not provide information as to which systems and organs might be affected by the limiting toxicant. Another major shortcoming of the LD_{50} test protocol is that all results are biased toward the short-range effects on the test animals (less than 14 days). Finally, with regard to a dynamic fire exposure, a noxious or toxic agent may exert a temporary disabling effect, which will prevent escape but will not in itself exert a lethal effect, thus, the animal is overcome by other agents and listed in the LD_{50} count.

The Survival Response Test Protocol: An animal conditioning chamber was designed and constructed which permits a greater degree of sophistication than was possible previously. This conditioning chamber utilizes a strobe-light warning of impending electrical stimulus. The test subjects are trained in an avoidance response utilizing a hind-leg flexion response mechanism (*32*). Figure 1 illustrates the operational experimental apparatus used to train the test specimens in the avoidance response. This avoidance response is being used to monitor the loss of survival responses during exposure studies.

The use of the sling restraining device in the conditioning and exposure studies maintains the subject in a convenient position for determination of respiratory rate, EEG, EKG,
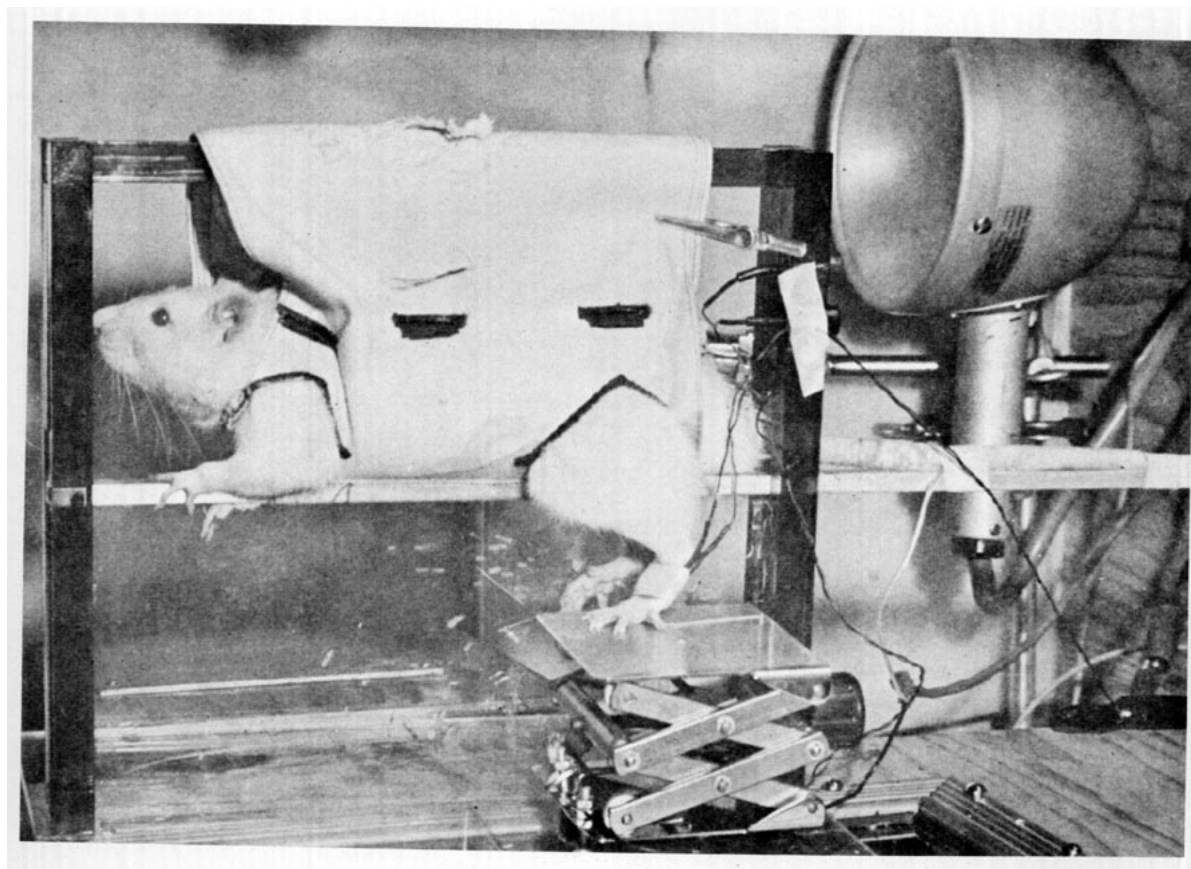

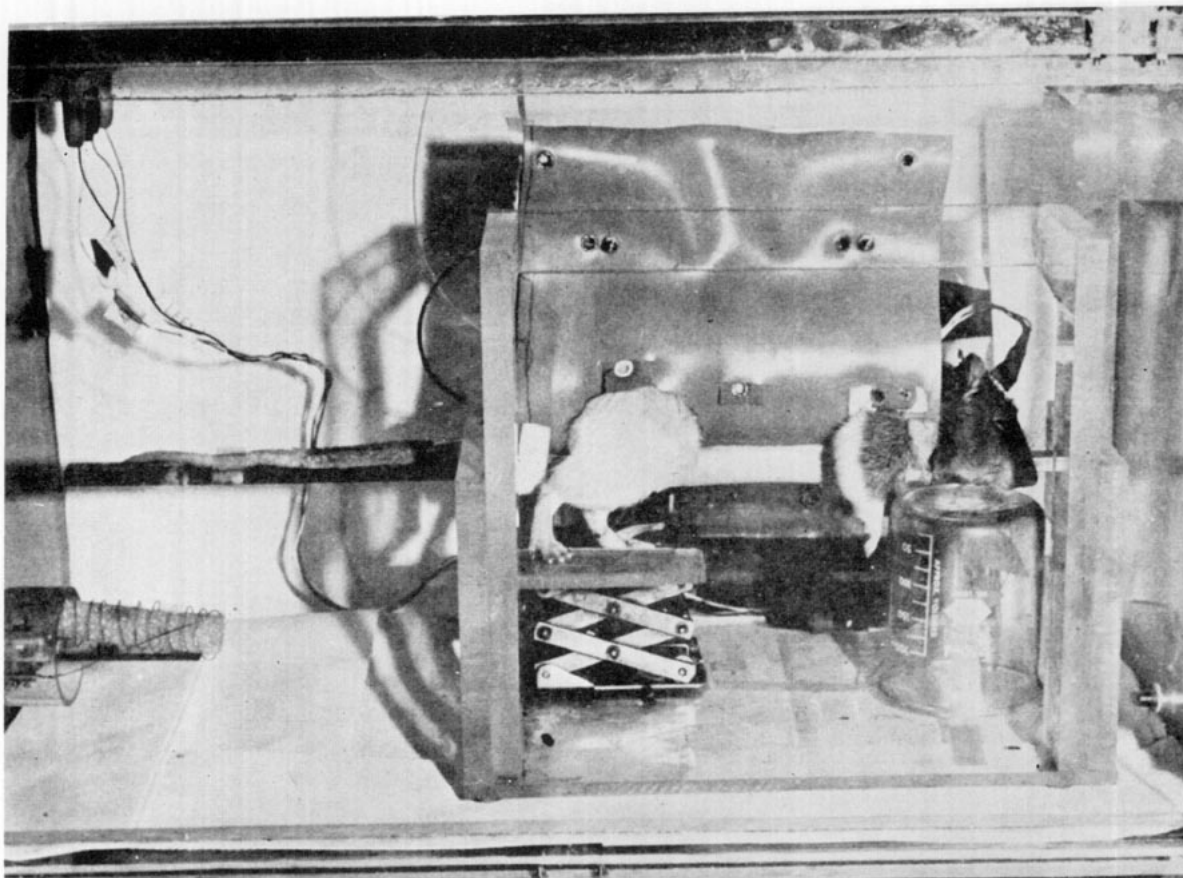FIGURE 1. Experimental set-up for animal chamber.

FIGURE 2. Animal model showing sling-type restrainer, conditioning module, and physiological recording leads.

peripheral nerve conduction velocity, reflex response latency, carboxyhemoglobin, body temperature, and operant behavior. An animal exposure chamber was appropriately modified to contain the sling and necessary connecting ports for physiological monitoring. The new system provides an animal model for investigation of the effects of a variety of noxious gases and a number of factors affecting animal response, such as age, drug action, altered body temperature, and repeated exposures.

A modified analytical procedure was developed by Petajan, Packham, and Frens (33) for determining carboxyhemoglobin in small volumes of rat blood as well as the development of a method which permits withdrawal of blood aliquots during actual exposure. It is now possible to withdraw blood samples during exposure chamber experiments (Fig. 2) for a variety of determinations including carboxy-

hemoglobin, hemoglobin, and oxyhemoglobin, electrolytes, serum enzymes, and various intoxicants.

The conditioned avoidance response has been modified so that it can be determined with the animal in the sling. The rat's rear leg is affixed to a lever which stabilizes and controls the direction of leg flexion and to which a weight can be added to compel the rat to exert force when withdrawing the leg. A metal plate that activates a relay lies beneath the foot, which when touched, delivers a shock to the rat's leg by means of a fine needle electrode inserted just beneath the skin of the leg. A strobe light provides a warning signal, which is followed in 5 sec by a shock. The warning signal and shock are delivered every 10 sec for a period of 1 min, with a 1-min rest period in between. Rats can be conditioned to avoid the shock in response to the warning light, that is receiv-

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ing only one reminder shock, in two sessions of 1 hr each. The conditioning chamber, previously described, has been built for training rats, and the necessary response counters are in operation. This response is now defined as the "survival response," the loss of which defines Level 2. Modification of this response can include changing of the warning stimulus mode (sound, touch, etc.), and the amount of work required to accomplish the flexion response.

Packham, Petajan, and Frens (33) have used the protocol described previously to monitor animal response to simulated conditions which may be encountered in "real fires." The following types of experiments have been carried out: determination of carboxyhemoglobin and vital functions when the survival response is lost; determination of influence of a given level of intoxication upon the acquisition rate of the survival response; determination of the loss of the learned survival response as a consequence of a given level of intoxication; determination of the alteration of nervous system function, such as peripheral nerve conduction velocity, spinal reflex activity, and cortical activity which accompany loss of the operant response.

In summary, the above experiments examined conditions responsible for the acute loss of the survival response, as well as its loss or impaired acquisition as a late sequela of carbon monoxide exposure.

Sensory and motor nerve conduction in the ventral caudal nerve has been determined *in vivo* in rats reaching Levels 3 and 4 by Petajan, Packham, and Frens (33). Fine 26-gauge needle monopolar electrodes were used for stimulating and recording the evoked muscle or nerve action potential. Initial experiments determined the change in conduction velocities following exposure to Level 3. At Level 3, carboxyhemoglobin ranged from 60% to 80%. The control conduction velocity was 26–28 m/sec. Animals exposed to Level 3 had a greater than 20% decrease in conduction velocity within the first hour after exposure. In 50% of the animals, the nerve became unresponsive after the first hour. Conduction remained significantly decreased for 24–48 hr, then tended toward normal, but remained eratic over the next 4 weeks. Sensory nerve conduction was most affected. An important observation of these experiments was the con-

tinued decrease in conduction following removal from the chamber. This effect is compatible with the observation that carbon monoxide migrates into muscles and other tissues from blood during hypovolemic shock (34). Animals exposed to Level 4 developed neuropathy similar to that seen at Level 3, but an additional effect was seen as well. Following the recovery period, neuropathy sometimes developed again as a late sequela.

Using the animal model, conduction velocity was determined during the course of exposure to carbon monoxide. In a second series of experiments, conduction velocity was followed through all levels to Level 4. The continued decrease in velocity following exposure to room air was observed. Follow-up studies were conducted every week in conjunction with detailed pathological studies of the nerves and other tissues.

The essential observation of this experiment was that attainment of Level 3 was essential for the development of latent pathology. At this level conditions prevail which allowed the "penetration" of carbon monoxide into tissues. Apparently metabolism was affected in such a manner that processes essential to cell function were impaired.

Pathologic Changes in Rats Exposed to Carbon Monoxide: Electron microscopy of the peripheral nerves have shown striking changes due to the effects of CO on rats. At Level 2 where the animal appeared to lose his survival response, gross and light microscopic pathological studies of brain, spinal cord, peripheral nerves, and internal organs of animals sacrificed immediately after removal from the chamber have shown no differences from control animals.

Level 3 was the level of motor collapse or loss of postural tonus. Ten of the rats at this level were studied grossly and by light microscopy 30 days after exposure. Brain, spinal cord, organs, and peripheral nerves showed no differences from control animals on light microscopy examination. However, changes were seen with the electron microscope at the nodes of Ranvier of peripheral nerves (also seen in animals exposed to Level 4). The anterior horn cells of the spinal cord showed no axonal chromatolysis or increased satellitosis.

In Level 4, the level of anoxic shock, all animals lost their ventral caudal nerve conduc-

tion. If the animals survived, maximal ventral caudal nerve conduction velocity returned, but motor potentials were small and polyphasic. These potentials disappeared again and returned to normal at 9–13 days after exposure. These findings suggested a delayed peripheral neuropathy such as has been reported in man. Since the delayed central nervous system lesions are demyelinative in character, it might have been expected that the peripheral nerve changes would be primarily demyelinative. However, this has not been borne out by our electron microscopic studies. The brain and other organs showed no differences from control animals grossly or with list microscopy. Some animals showed axonal chromatolysis of some small anterior horn cell but no increased satellitosis. The only lesions seen in peripheral nerves with light microscopy were an increase in mast cells and some areas of vacuolated myelin. No decrease in axons or myelin sheaths was evident. With the electron microscope, the damage to the peripheral nerve was seen primarily at the node of Ranvier and in the Schwann cell after exposure to Levels 3 or 4.

When rats are exposed to the "anoxic shock" level of carbon monoxide intoxication, they develop a carboxyhemoglobin level of 60–80%. Clinically these animals show a decrease in blood pressure, decreased and irregular respiration, and cardiac arrhythmias. They also show a decrease in ventral caudal nerve conduction velocity, usually to zero. If removed from the CO environment at this time they survive. The ventral caudal nerve conduction velocity returns to normal in from 2 to 8 days. Sensory conduction is most impaired. Although animals show no weakness during recovery, they are hypersensitive to stimuli.

Ventral caudal and peroneal nerves from rats exposed to the anoxic shock level of CO intoxication were examined 7, 10, 14, 21, and 28 days after exposure. Light microscopy of the nerves did not reveal any abnormalities to account for the decreased conduction velocity. However, with electron microscopy changes were seen at the node of Ranvier, both in large and small myelinated nerve fibers. The changes were more severe in large myelinated fibers than they were in small fibers.

The speed of progression of damage and repair depended on the size of the fiber and somewhat on the individual animal studied, but
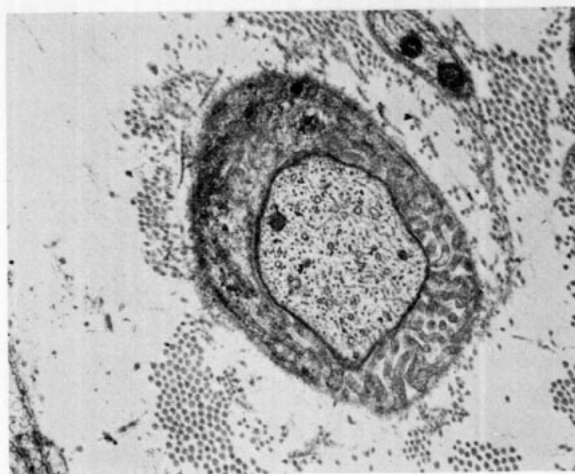

FIGURE 3. Normal nodes of Ranvier showing fingerlike processes of Schwann cell extending toward the axoplasm. Cross section, 8960×.

the steps in the progression appeared the same for large and small fibers. Most, though not all, nodes showed these changes.

Normally a thin sheet of cytoplasm from the Schwann cells on either side of the node or fingerlike processes from the Schwann cells make contact at the center of the node. Each Schwann cell also sends fingerlike processes vertically toward the axolemma (Fig. 3). After exposure to CO the major portions of cytoplasm from the Schwann cells from both sides of the node appear to retract and disengage. The many fingerlike processes also appear to retract into the main mass of Schwann cell cytoplasm. The myelin terminals may remain attached to the axolemma in small fibers. The myelin lamellae are stripped away from one another. The myelin terminals and adjacent myelin are completely or partially destroyed in many large fibers, with myelin figures appearing in the Schwann cell cytoplasm and axoplasm.

Figure 4 shows that the first-formed myelin terminals are sometimes preserved. At the time of greatest destruction the axon is frequently covered by only a thin sheath of Schwann cell cytoplasm and basement membrane or basement membrane alone. The axoplasm at this point is swollen. In larger diameter fibers, microtubules and neurofilaments are disrupted in this area as well (Fig. 5). Large-sized fibers are most severely damaged.
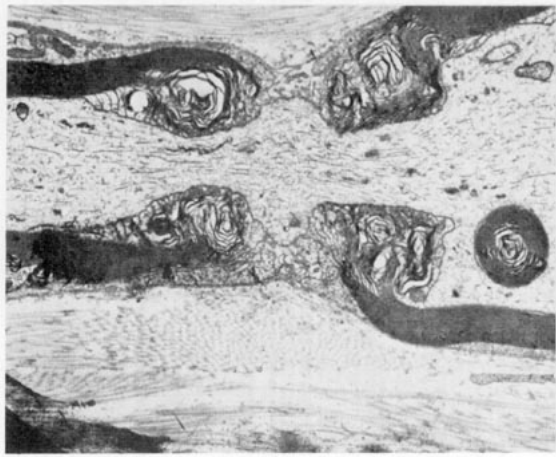
FIGURE 4. Node of Ranvier, 7 days after exposure to anoxic shock level of CO poisoning, showing myelin degeneration and retraction of myelin terminals and Schwann cell cytoplasm and processes. Longitudinal section, 3360×.
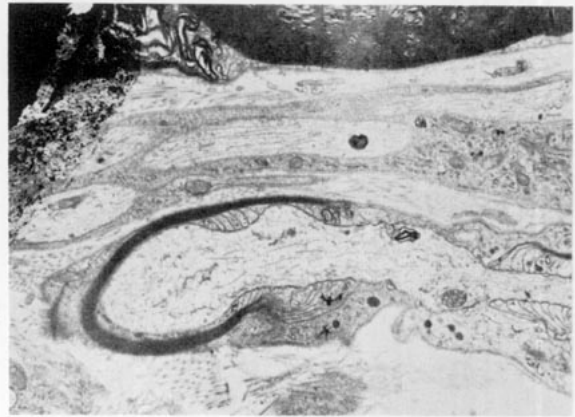

FIGURE 6. Node of Ranvier, 14 days after exposure to anoxic shock level of CO poisoning, showing elongation of node and covering of the axon by swollen Schwann cell processes. Longitudinal section, 2940×.
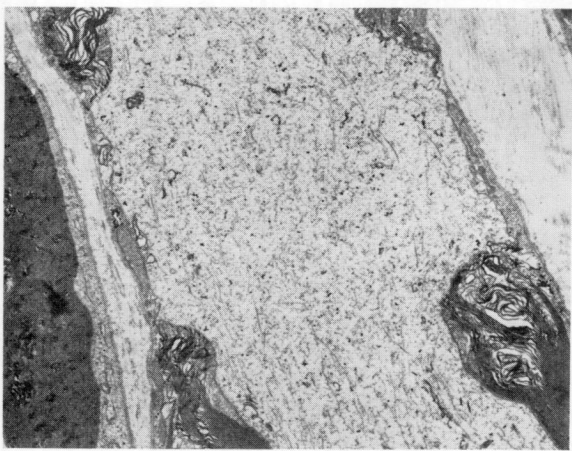

FIGURE 5. Node of Ranvier, 7 days after exposure to anoxic shock level of CO poisoning, illustrating loss of Schwann cell processes, swelling of axon, and loss of covering axoplasm. Longitudinal section, 3120×.
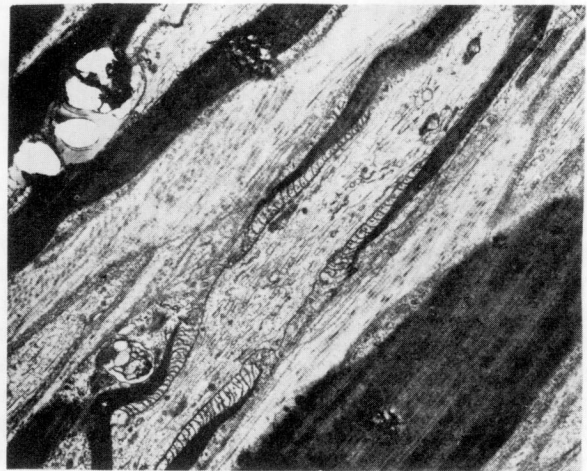

FIGURE 7. Node of Ranvier, 21 days after exposure to anoxic shock level of CO poisoning, showing return of fingerlike processes but an elongated node and myelin debris in the Schwann cell. Longitudinal section, 3120×.

From 7 to 10 days after exposure, the process of destruction seems almost complete in large myelinated fibers but is still going on in smaller fibers. The beginning of reparative changes is late in larger diameter fibers. The beginning of reparative changes is late in larger diameter fibers, still being incomplete at 28 days. In smaller nerve fibers repair is often beginning by 14 days, although it too is not complete at

28 days. The first step in repair appears to be swelling of the Schwann cell cytoplasm or production of processes to cover the bare node (Fig. 6).

From 14 to 28 days the fingerlike projections of Schwann cell cytoplasm begin to appear at the node (Fig. 7).

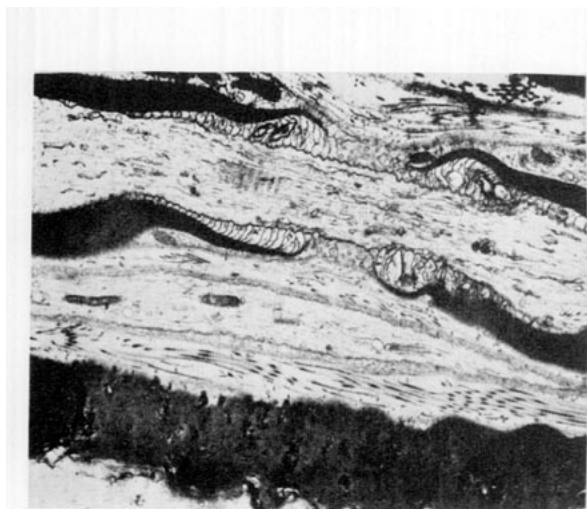Complete joining of thin layers of Schwann cell cytoplasm or fingerlike processes from

FIGURE 8. Node of Ranvier, 28 days after exposure to anoxic shock level of CO poisoning showing nearly normal Schwann cell processes, although myelin debris still remains. Longitudinal section, 3120×.
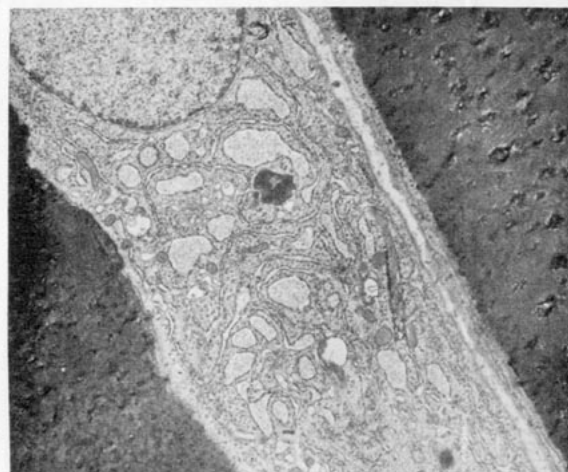

FIGURE 9. Schwann cell cytoplasm, 14 days after exposure to anoxic shock level of CO poisoning, showing marked dilatation of cisternae of rough endoplasmic reticulum with evidence of protein synthesis. Longitudinal section, 3360×.

either side of the node overlying vertically-oriented fingerlike processes is not often seen (Fig. 8).

Where joining is seen, there is often also reduplication of Schwann cell basement membrane over the node. The total process of reconstitution of destroyed myelin terminals and Schwann cell structures at the node was not observed. The node is rarely completely repaired 28 days after exposure. In smaller fibers, which showed less nodal damage, myelin terminals tend to appear normal, with some myelin debris in the Schwann cell cytoplasm, but nodes still appear elongated. Repair of myelin at the node occurs after Schwann cell repair. Nerves from exposed rats before 7 and after 28 days must be examined before the entire process is clear. Only a few macrophages or inflammatory cells were seen in any nerves.

At 7 days after exposure to CO, Schwann cells show some dilatation of smooth and rough endoplasmic reticulum with increased density and occasional vacuolization of mitochondria. These changes are more striking 14 days after exposure, at which time, evidence of protein synthesis within the dilated cysternae of the rough endoplasmic reticulum is seen (Fig. 9). These changes could still be seen 28 days after exposure.

Following exposure to CO a few unmyelinated axons exhibit swelling and loss of internal structure, but most appear normal.

In our experiments we have studied the brains and spinal cords of rats exposed to the anoxic shock level of CO intoxication at various times after exposure by light microscopy only. Six animals were studied 7 days after exposure, two animals 14 days after exposure, two animals 21 days after exposure, nine animals 28 days after exposure and four animals 2 months after exposure. There was no apparent loss of neurons from the central nervous system grey matter or demyelination or necrosis of white matter in any animal. However, exposed rats did show difficulty in learning a conditioned response 30 days after exposure when compared with control rats. This is evidence that CO probably produced some central nervous system damage.

Recently, Miyagishi (35) has shown electron microscopic lesions in the cells of the white and grey matter of rats exposed acutely and chronically to CO. These consisted of marked dilatation of the endoplasmic reticulum of the oligodendroglia, the central nervous system equivalent of the Schwann cell. Abnormalities of some myelin sheaths were also noted. Neurons showed some fragmentation or dilatation of rough endoplasmic reticulum and golgi, but no mitochondrial changes.

Comparison of the Results of Carbon Monoxide Intoxication in Rats and Humans: It may be asked what these lesions in the peripheral nerves of the rat have to do with the

nervous system changes produced by CO in the human. Peripheral neuropathies occur in humans following CO exposure but are thought to be rare (36,37). However, they may be more common than is thought, since the neuropathy may be mild and fleeting, with central nervous system manifestation so severe that the neuropathy is overlooked. Also, the rats rarely showed severe clinical evidence of neuropathy; similarly, affected humans might not notice any neurological deficit.

The most common human consequences of CO poisoning are seen in the central nervous system if the patient survives long enough. This consists of loss of neurons, astrocytosis, and sometimes necrosis of the globus pallidus, cerebral cortex, hippocampus and Purkinje cells of the cerebellum. There is variation in pathology not completely related to the degree or length of exposure to CO. About 10% of patients who survive show "pseudorecovery," that is, they appear to awaken from coma and normalize, only to deteriorate mentally and neurologically weeks or months later (38). This is usually due to delayed demyelination and necrosis of the central nervous system white matter.

Recently we encountered a 21-year-old patient who was exposed to CO for an unknown period of time to an unknown CO-Hb level. He was admitted to the hospital in a comatose condition and remained in coma for almost a month. Following this, he began to regain function. Approximately a year after his admission he was able to return to college, although he had residual spasticity of the lower limbs, some inappropriateness and emotional lability. He drowned while scuba diving 18 months after admission. At autopsy the brain showed evidence of possible loss of neurons only in the globus pallidus, with other grey structures appearing intact. There was loss of myelinated fibers from the long tracts of the spinal cord and active demyelination of the cerebral white matter. The cause of the demyelination in CO poisoning is unknown, though it is well known that cyanide and hypoxia can also produce necrosis of white matter in primates and humans (39–43).

Carbon Dioxide

All fires will produce some levels of carbon dioxide which, in turn, may be inhaled by those in the vicinity of a fire. Since CO_2 is an important constituent of the body process, CO_2 is not considered a toxic agent at normal concentrations. Inhalation of carbon dioxide will, however, stimulate respiration which, in turn, will increase inhalation of possible toxic components from the combustion and noncombustion gases present from the fire. It is not correct to assume, however, that toxic signs and symptoms will not occur in man. For example, inhaling of CO_2 in concentrations of 10% have caused headaches and dizziness, as well as other symptoms in segments of test groups. Higher concentrations (above 20%) can lead to narcosis in animals and in most people.

Sulfur Dioxide

Certain natural materials, as well as manmade materials such as natural and synthetic rubbers, may have sufficient sulfur content to generate sulfur dioxide directly or indirectly when the materials are exposed to heat and fire. This gas (SO_2) is a pungent, heavy gas and is extremely toxic to animals and humans (44). The threshold limit value (TLV), i.e., the concentration as a weighted average over an 8-hr period of a compound in the air which, if exceeded, may cause toxic signs and symptoms, is given as 5 ppm. Sulfur dioxide, in contact with water (moisture), will form sulfuric acid which, in turn, produces the extremely irritant response when the gas has contact with skin. Mucous membranes, in particular in the respiratory tract and in the eye, are highly susceptible to the irritant effects. Exposure to high concentrations of the gas lead to death, most likely because of asphyxiation (blockage of air transport in the upper respiratory tract). Chronic exposure to sulfur dioxide appears to have greater toxic effects upon those having cardiorespiratory diseases than those not suffering with these ailments. Epidemiologic studies have also led to suggestions that a cause–effect relationship may exist for the high incidence of death during episodes of smog.

Hydrogen Cyanide

Hydrogen cyanide is produced in varying concentrations from many nitrogen-containing

organic compounds during pyrolysis or combustion. Since hydrogen cyanide is a highly endothermic compound, its concentration will increase as the temperature of combustion rises and may form as the result of nitrogen fixation processes.

Cyanide reacts with Fe (III) of the cytochrome oxidase in the body, thus preventing the utilization of oxygen by the tissue (blocking of intracellular oxygen transport).

Concentrations above 20 ppm in the air are considered as dangerous to health. Initial inhalation of HCN vapors will cause a reflex stimulation of breathing which, in turn, will lead to greater concentrations of the gas entering the body. The cause of death is paralysis of the respiratory center of the brain. Table 9 presents a summary of symptoms in humans in relation to the HCN concentrations in the air (45).

Hydrogen Chloride

Degradation of poly(vinyl chloride) produces hydrogen chloride as one of its major degradation products. On combining with water, hydrogen chloride forms hydrochloric acid. This compound will cause destructive damage to mucous membranes. If inhaled, the upper respiratory tract will be severely damaged and this may lead to asphyxiation and death. Tables 10 and 11 present a summary of the toxicological effects to HCl to humans and animals (45).

Aliphatic Hydrocarbons

Thermodegradation of all organic polymers will produce a variety of aliphatic compounds (44) having a range of molecular weights. The lower molecular weight compounds will

Table 9. Relation of hydrogen cyanide concentrations in air and symptoms in humans.

HCN concentration, ppm	Symptoms
0.2-5.0	Threshold of odor
10	(TLV-MAC)
18-36	Slight symptoms (headache) after several hours
45-54	Tolerated for 1/2-1 hr without difficulty
100	Death—1 hr
110-135	Fatal in 1/2-1 hr
181	Fatal in >10 min
280	Immediately fatal

Table 10. Effects of inhalation of hydrogen chloride on humans.

Hydrogen chloride concentration in air, ppm	Symptoms
1-5	Limit of odor
5-10	Mild irritation of mucous membranes
35	Irritation of throat on short exposure
50-100	Barely tolerable
1000	Lung edema after short exposure

Table 11. Inhalation toxicity of hydrogen chloride on animals.

Hydrogen chloride concentration in air, ppm	Species	Effects
50	Monkeys	Tolerable for 6 hr daily
300	Guinea pigs	Mild corneal damage after 6 hr
3,200	Mice	No mortality after 5 min
4,300	Rabbits	Lung edema, death after 30 min
13,745	Mice	$LD_{50} = 5$ min
30,000	Rats	No mortality in 5 min
41,000	Rats	$LD_{50} = 5$ min

Table 12. Maximum allowable concentrations (MAC) of common aliphatic hydrocarbons.

Compound	MAC, ppm
Propane	1000
Butane	1000
Hexane	500
Heptane	500
Octane	500
1,3-Butadiene	1000

produce narcosis in animals and man but the biological activity decreases on going to higher members of the series. With certain polymers unsaturated hydrocarbons may be present when the polymer is degraded which will generally have a greater toxic effect than the corresponding saturated compounds. In these mixtures there may also be present acids, alcohols, and aldehydes, each contributing a toxic property. Table 12 summarizes the maximum allowable concentrations of common aliphatic hydrocarbons (44).

Aromatic Hydrocarbons

Aromatic hydrocarbons (44) starting with benzene and leading to other aromatic structures, will have irritating properties as well as systemic toxicity. As the structure of the aromatic molecule is altered, the toxicity may be increased or decreased. Several aromatic compounds, such as benzene, may be absorbed not only by inhalation but also by absorption through the skin. Levels of 100 ppm and above are considered dangerous to health. Styrene is a depolymerization product of polystyrene and is considered as safe in concentrations below 100 ppm. Levels above 100 ppm can produce irritation to mucous membranes, symptoms of toxicity, and impairment of neurological functions.

Acrolein, Formaldehyde, Acetaldehyde, Butyraldehyde

Acrolein, formaldehyde, acetaldehyde, and butyraldehyde are examples of low molecular weight compounds which are commonly found in the smoke from pyrolysis or combustion of plastic materials. Acrolein is a three-carbon compound possessing the chemical formula CH_2CHCHO. This compound, due to its extreme lachrymatory effect, serves as its own warning agent. It affects particularly the membranes of the eyes and respiratory tract. The maximum allowable concentration (MAC) is 0.1 ppm. Table 13 gives concentrations of acrolein and other toxic aldehydes found in the smoke released during the combustion of several common materials (22).

Zikria pointed out in a recent paper (22) that carbon monoxide alone cannot account for the pulmonary edema and tracheobronchial and pulmonary parenchymal damage resulting from

Table 13. Smoke analyses on combustion of several common materials.

Compound	Concentration, ppm (volume)			MAC[a]
	Wood	Kerosene	Cotton	
Acrolein	50	<1	60	0.1
Formaldehyde	80	<10	70	5.0
Acetaldehyde	200	60	120	200
Butyraldehyde	100	<1	7	Not tested

[a] Industrial maximum allowable concentration.

smoke poisoning. He exposed dogs to standardized smokes of wood and kerosene, without heat, in a smoke chamber. The animals exposed to kerosene smoke did not have any pulmonary edema, tracheobronchial or parenchymal damage, and all survived. On the other hand, the animals exposed to wood smoke did develop pulmonary edema and tracheobronchial and parenchymal damage, 50% of the test animals dying with 1-3 days after exposure.

Deichmann and Gerarde (46) reported that acrolein in a concentration of 5.5 ppm has been shown to cause irritation of the upper respiratory tract; at higher concentrations, pulmonary edema occurs; and at concentrations of 10 ppm, death occurs within a few minutes. Sim and Pattle (47) subjected human volunteers to acrolein. They reported that inhalation of acrolein causes lacrimation and irritation of all exposed mucous membranes at concentrations of as little as 0.805 ppm.

Synergistic Effects During Fire Exposure

Recent investigations by personnel of the Applied Physics Laboratory, The John Hopkins University and the Flammability Research Center of The University of Utah, have determined that a strong synergistic effect may occur as the result of inhalation of the products of combustion of natural and synthetic materials by an individual under the influence of alcohol. Approximately 85% of those fire deaths involving cigarettes and mattresses or upholstered furniture have been traced to individuals who fell asleep while smoking and under the influence of alcohol. Laboratory analysis of the blood of these victims indicates that the carbon monoxide saturation is typically below 40% when the individual has a blood alcohol level 0.15 mg-% or greater. Several cases have been studied where blood alcohol levels exceeding 0.4 mg-% were measured. In all of these cases the carboxyhemoglobin saturation was less than 25%. Reconstruction of the fire scene as well as information gathered during test burns of selected mattresses and upholstered furniture in room and corridor tests have indicated that a period of 3-5 hr is required before the onset of flaming ignition. However, inasmuch as the victim falls asleep and the ingnition source, the cigarette, is permitted to come into contact with the surface on which the individual

is sleeping, the victim is then subjected to a highly localized concentration of carbon monoxide and other toxic or noxious gases during the smoldering period before the onset of flaming combustion.

Similar synergistic effects may play a major factor in human survival during fire exposure. Possible synergistic effects may be attributed to the levels of oxygen, carbon dioxide, carbon monoxide, aldehydes, ketones, alcohols, acids, and the temperature level to which fire victims are exposed. Further research is required to determine on a quantitative basis, the exact contribution of each of the above classes of toxicants.

Analysis of Combustion Products

As discussed previously, the products produced during the combustion of natural and synthetic materials are many and varied. Some of the degradation products are produced in copious amounts, while others may be produced in trace quantities that render detection extremely difficult. Additional complications manifest themselves in potential cross-reactions resulting from products of decomposition which may result in a very short half-life for the original products.

It should be noted that such factors as the density, geometry, altitude, and surface area of the material, as well as many physical factors which include but are not limited to environment, rate of heating, energy flux, presence of a pilot flame, degree of ventilation, development of a char structure, can greatly influence the mechanism of decomposition and the products resulting from the decomposition process. Within the scope of this paper, three separate and distinct processes will be considered, i.e., pyrolysis, oxidative degradation, and flaming combustion.

Computerized Analytical System

Two computerized analytical systems are employed in the Flammability Research Center: a gas chromatograph/quadropole (electron impact) mass spectrometric system and a gas chromotograph/chemical ionization mass spectrometer system.
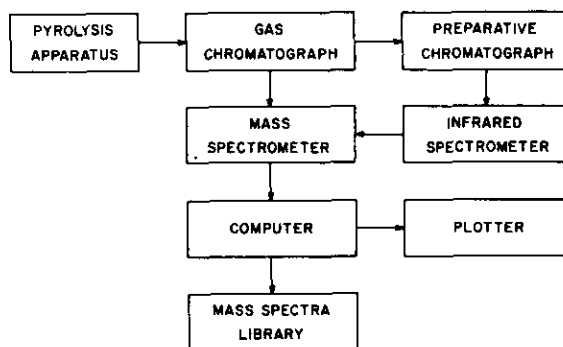
The computerized analytical system utilizing

FIGURE 10. Schematic representation of computerized analytical system.

the quadropole mass spectromer is shown schematically in Figure 10.

Samples may be introduced into the analytical system as gases, liquids, or solids. A brief description will be given here for the pyrolysis procedure using the solid pyrolysis probe. Weighed samples are placed in the pyrolysis probe and pyrolyzed directly into the inlet of the gas chromatograph. A delayed mode is used during which time, the sample was permitted to reach equilibrium with its environment. Pyrolysis temperatures ranging from 300 to 1000°C are obtained by varying the amperage supplied to the filament probe. A 6-in. stainless-steel insert, filled with glass wool, is connected as a precolumn filter to prevent large particles from reducing the flow rate of the effluents. A research gas chromatograph, capable of subambient operation, was employed for the separation of the decomposition products. The system has available three detectors, a thermal-conductivity detector (TCD), a flame-ionization detector (FID), and an electron-capture detector (ECD). Any two of these detectors can be used simultaneously by using a splitter. For the results reported here, the thermal conductivity detector and flame ionization detector were used. A splitting ratio (TCD to FID) of 10 to 1 was employed.

A list of columns commonly employed to separate the products resulting from thermal decomposition of natural and synthetic materials is presented in Table 14. Within the scope of this paper, the products of thermal decomposition, principally low molecular weight gases, are separated by using a $\frac{1}{8}$-in. diameter, 16-ft stainless-steel (SS) column with Chromosorb 101 packing. The higher molecular weight

Table 14. Gas chromatographic columns selected for analytical studies.

Column	Specification	Gas species
Porapak Q⎫ Porapak R⎭	8 ft×1/8 in., 80/100, ss	H_2, N_2, O_2, Ar, CO, NO, N_2O, CH_4, CO_2, H_2O, H_2S, SO_2, C_2H_6, COS, HCN
Chromosorb 101	8 ft×1/8 in., 80/100, ss	Glycols, alcohols, free acids
Chromosorb 103	8 ft×1/8 in., 80/100, ss	Amines
10% SP-1000 on washed Chromosorb W	8 ft×1/8 in., 100/120, ss	Alcohols, free acids
20% DEGS on Chromosorb W (AW)	8 ft×1/8 in., 80/100, ss	C_8-C_{24} fatty acids
10% Apiezon L wax on Chromosorb W (HP)	8 ft×1/8 in., 80/100, ss	Hydrocarbons— aromatics
10% Carbowax 20M on Chromosorb W (HP)	8 ft×1/8 in., 80/100, ss	Alcohols, ketones, acetates, essential oils
Tenax GC	8 ft×1/8 in., 80/100, ss	High boiling polar compounds, alcohols, poly(ethylene glycol) compounds, diols, phenols, mono- and diamines, ethanolamines, amides, aldehydes, and ketones
OV-1	8 ft×1/8 in., 80/100, ss	Hydrocarbons
3% Dexsil 300 GC	8 ft×1/8 in., 100/120, ss	High temperature hydrocarbons

degradation products are separated by using a ⅛-in. diameter 8-ft stainless steel Dexsil column.

An automatic electronic integrator with printout facilities is used to record chromatographic peak areas and retention times for quantitative analysis. Internal standards are injected into the gas chromatograph in varying amounts to produce a plot of standard weight versus peak area. A table of response factors for gas chromatographic analysis (48) is used to calculate the amounts of products obtained from the thermal degradation of the materials being studied.

The identification of materials separated by the gas chromotograph is obtained by using a modified quadropole (known as a dodecapole) mass spectrometer. This system completely scans a mass range of 1 to 600 amu in 2 sec. This is fast enough to permit spectral identification of separate GC peaks in real time. In the periodic scan computer-controlled mode, the mass spectrometer repetitively scans a complete GC run and stores all of the data on a disk.

The use of computers for the interpretation of chemical spectra has been covered thoroughly in the literature (49–57). The actual search proceeds through a comparison of the unknown spectra with the complete set of library spectra on some selected subset of these spectra. The selection of the subset usually involves some known characteristics of the unknown compound, such as molecular weight, empirical formula, etc.

The principal aim of the search routine is to relate known spectra which "best fit" the unknown, according to a preselected criterion.

Conventional mass spectrometry techniques (electron impact ionization) often impart enough energy to the ions formed that fragmentation is so extensive as to preclude sample identification. When this presents a problem, chemical ionization techniques may be used.

Chemical ionization (CI) mass spectrometry is a technique first described by Field et al. (58) in which ions are produced by chemical reactions in the gas phase, rather than by impact of energetic electrons. The technique is carried out by allowing an electron beam to impinge on a reagent gas at a pressure of approximately 1 torr. Ion–molecule reactions will take place in this environment to produce electronically and thermally deactivated ions which may then react with the species under investigation. Since the cross section for ion–molecule reactions is greater by a factor of 50 or more than that for electron-impact reactions and the material under analysis is present in low concentration (approximately 0.01%), nearly all ionization of the sample is produced by proton-transfer reactions. These "chemical" reactions are of considerably lower energy than reactions involving ionization by electron impact. Typically, the energies liberated in proton-transfer reactions are 5–20 kcal/mole, depending on the reagent gas used. When the ionization energy is of this low magnitude, typically only pseudo-parent ions (ions having gained or lost a proton) are formed, and decomposition of these ions does not take place. The result of tert-butyl ions (from isobutone reagent, with a 5 kcal/mole transfer energy) reacting with alkanes, alkenes, drives, etc., produce pseudo-parent ions by a hydride abstraction mechanism. The CI method becomes the method of choice for

determining the distribution of compounds in a complex mixture, since it produces with isobutane reagent only pseudo-parent ions with no fragmentation.

Analysis of Decomposition of a Urethane Polymer

Hileman et al. (59) have recently described the use of the computerized analytical system to qualitate and quantitate the products resulting from pyrolysis of a flexible-urethane foam. A brief review of this report is presented to illustrate the techniques employed. A 2–3-mg portion of polymer was pyrolyzed at temperatures ranging from 300°C to 1000°C. The pyrolysis products were separated on a variety of columns including Chromosorb 101, Chromosorb 103, Porapak Q, OV-1, Dexsil 300 GC, and Carbonax 2012. The column temperature was controlled between 0 to 200°C at a program rate of 10°C/min. Identification of the products

was obtained from the tabulation of the corresponding mass spectrum of a particular GC peak. To aid in identification, the Aldermaston library of mass spectra data was available for computer searching. As an additional aid, retention indices based on the principle of Kováts (60,61) were determined for all compounds on the columns used. Thus, the combination of retentive index and mass spectral data allowed identification of all major pyrolysis products. Quantitative analysis of the pyrolysis products was facilitated utilizing the peak area recorded by the interfaced electronic integrator. The peak areas were corrected for response factors (48) and referenced to ethanol as an internal standard for quantification.

Figure 11 presents a typical chromatogram (59) obtained from the pyrolysis of the flexible urethane foam with the major peaks numbered and the identification of these peaks given in Table 15 (59). Table 15 also shows the method of identification for each peak.
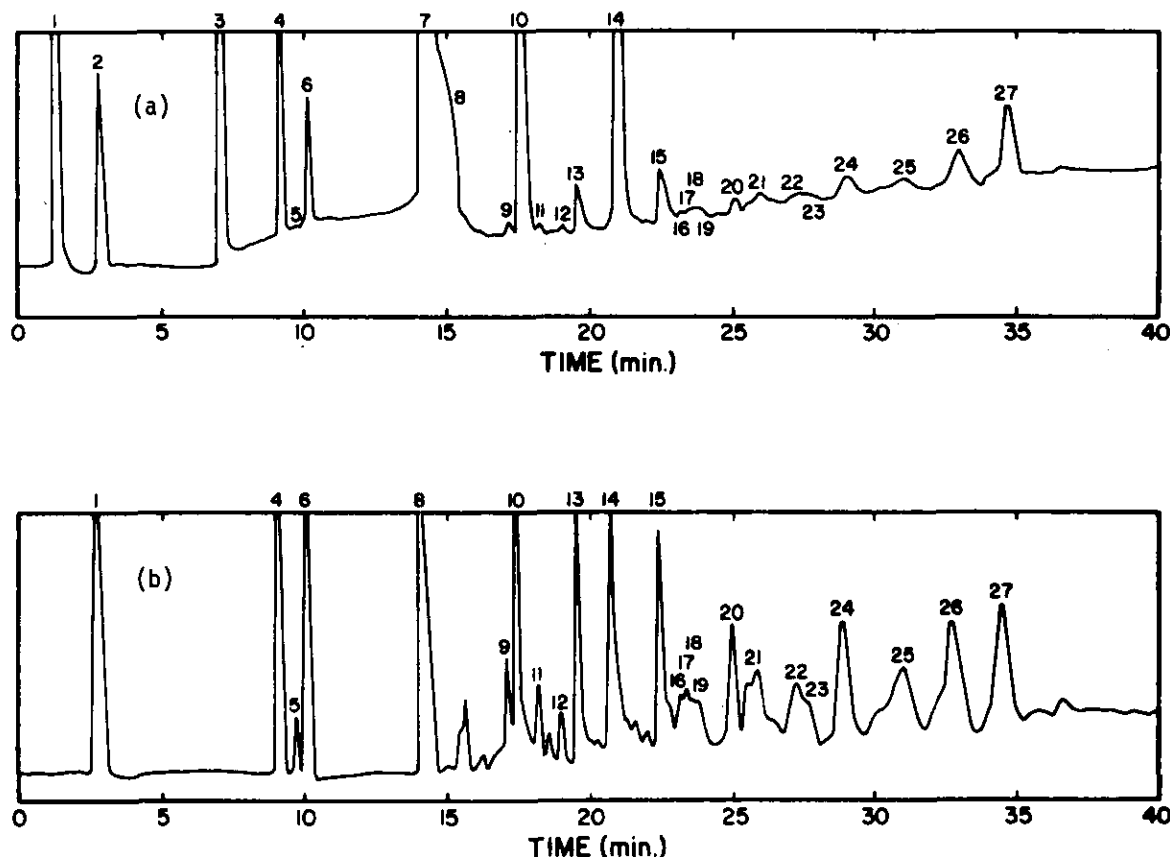

FIGURE 11. Chromatograms of the products of thermal decomposition (1000°C) of a flexible-urethane foam on Chromosorb 101: (a) thermal conductivity detector (TC); (b) flame ionization detector (FID).

Table 15. Peak identification for thermal decomposition products resulting from flexible urethane foam.

Peak	Name	Method[a]
1	Carbon monoxide	GC/MS
2	Methane	GC/MS
2A	Nitrous oxide	GC/MS
3	Carbon dioxide	GC/MS
4	Ethene	GC/MS
5	Ethyne	GC/MS
6	Ethane	GC/MS
7	Water	GC/MS
8	Propene	GC/MS
9	Methanol	GC/MS
10	Ethanal	GC/MS
11	NI[b]	
12	Dimethyl ether	GC/MS
13	Ethanol	GC/MS
14	Propanal and acetone	GC/MS
15	Propanol	GC/MS
16	NI	
17	2-ethoxyethanol	MS
18	NI	
19	NI	
20	NI	
21	NI	
22	NI	
23	NI	
24	NI	
25	NI	
26	2-Isopropoxy-1-propanol	MS
27	Di-n-propoxymethane	MS

[a] GC indicates confirmation by use of Kováts' indices; MS indicates confirmation by mass spectral data.
[b] NI = not identified.

Table 16. Products obtained during the pyrolysis of flexible-urethane foam.[a]

Product	Concentration of product, %			
	300° C	500° C	750° C	1000° C
CO	—	—	0.19	1.65
CH₄	—	0.06	0.41	1.74
CO₂	0.21	0.55	0.13	1.12
C₂H₄	—	0.02	0.62	1.49
C₂H₂	—	—	—	0.05
C₂H₆	—	0.04	0.68	0.45
H₂O	0.10	—	0.55	0.47
C₃H₆	0.08	1.38	0.47	5.35
CH₃OH	—	—	0.02	0.06
CH₃CHO	Trace	0.71	1.86	4.75
CH₃CH₂OH	—	0.05	0.11	0.17
CH₃CH₂CHO	Trace	0.47	5.46	10.40
CH₃CH₂CH₂OH	—	0.08	0.19	0.38
TDI[b]	0.23	0.63	0.28	0.41

[a] Data obtained on Chromosorb 101 column unless otherwise indicated.
[b] Data obtained from a 3% Dexsil column.

Table 16 presents a summary of the quantitative analysis (59) of pyrolysis products obtained from the flexible urethane foam when it was subjected to temperatures ranging from 300 to 1000°C.

The major constituents eluted from the Chromosorb 101 columns are oxygen-containing compounds which result principally from the breakdown of the polyol. These conclusions were confirmed by the pyrolysis of the pure polyol. In order to insure that nitrogen-containing compounds were being irreversibly absorbed onto the chromosorb 101 column, Chromosorb 103, a porous polymer designed to separate nitrogen-containing compounds, was used. It should be noted that all major peaks observed on Chromosorb 101 were also observed on the Chromosorb 103 column. Table 17 presents compounds obtained by using the Chromosorb 103 column (59). Several nitrogen-containing compounds, such as propanitrile, methylpyridine, and benzonitrile which did not elute with the Chromosorb 101 column were observed.

Pyrolysis and Combustion Studies

Chemical analysis of degradation products

Table 17. Peak identification for thermal decomposition products resulting from flexible urethane foam.

Peak	Name	Method[a]
1 and 2	CO/CH₄	GC/MS
3	CO₂	GC/MS
4	Ethene and ethyne	GC/MS
5	Ethane	GC/MS
6	Propene	GC/MS
7	Propyne	GC/MS
8	Allene	MS
9	1-Butene	GC/MS
10	Ethanal + 1,3-butadiene	GC/MS
11	1-Butene-3-yne	MS
12	NI[b]	
13	NI	
14	NI	
15	NI	
16	Propanal	GC/MS
17	Acetone + 3-pentene-1-yne	GC/MS
18	Propenitrile	GC/MS
19	Propanitrile	GC/MS
20	NI	
21	Hexatriene	GC/MS
22	Benzene	GC/MS
23	Toluene	GC/MS
24	NI	
25	Methylpyridine	GC/MS
26	NI	
27	m-Xylene	GC/MS
28	Styrene	GC/MS
29	Benzonitrile	GC/MS
30	Indene	MS
31	Dicyanobenzene	MS

[a] GC indicates confirmation by using Kováts' indices; MS indicates confirmation by using mass spectral data.
[b] NI = not identified.

resulting from the combustion of materials is extremely complex, inasmuch as numerous degradation processes may be encountered simultaneously. In addition, certain effluents may serve to autocatalyze or retard the degradation of materials undergoing decomposition. In the dynamic fire environment there is often a rapid heat build-up which is greatly influenced by the presence of an excess supply of oxygen. Under some conditions the degradation products may also provide a fuel-rich environment, and this affects the combustion process. In an attempt to develop a model which will permit simulation of the fire environment, laboratory studies encompassing pyrolysis, oxidative degradation, and flaming combustion have been carried out. Figure 12 presents chromatograms obtained during laboratory-scale pyrolysis and combustion experiments involving the flexible urethane foams. Initial examination of the chromatograms presented in this figure showed definite changes in the nature of decomposition products. Of particular significance is the peak identified as hydrogen cyanide in the combustion chromatograph that

does not appear in the pyrolysis chromatogram.

Considerable additional research is required to determine whether many of the potentially noxious or toxic products produced as a consequence of pyrolysis or combustion may contribute to loss of the survival response and death or whether under certain conditions many of these products are actually destroyed themselves by cross reactions or actual flame exposure.

Effect of Fire Retardants

The importance of fire retardants to the overall growth of the plastics industry has led to the introduction of many new fire-retardant compositions with increasing frequency during the past decade. Although this expanding research in the field of fire retardation of polymeric materials has uncovered many new facts about the mechanism of the degradation process, the technology of fire retardancy in polymer compositions still retains a high degree of empirical character.

1. CO
2. CH_4
3. CO_2
4. N_2O
5. C_2H_4
6. C_2H_2
7. C_2H_6
8. H_2O
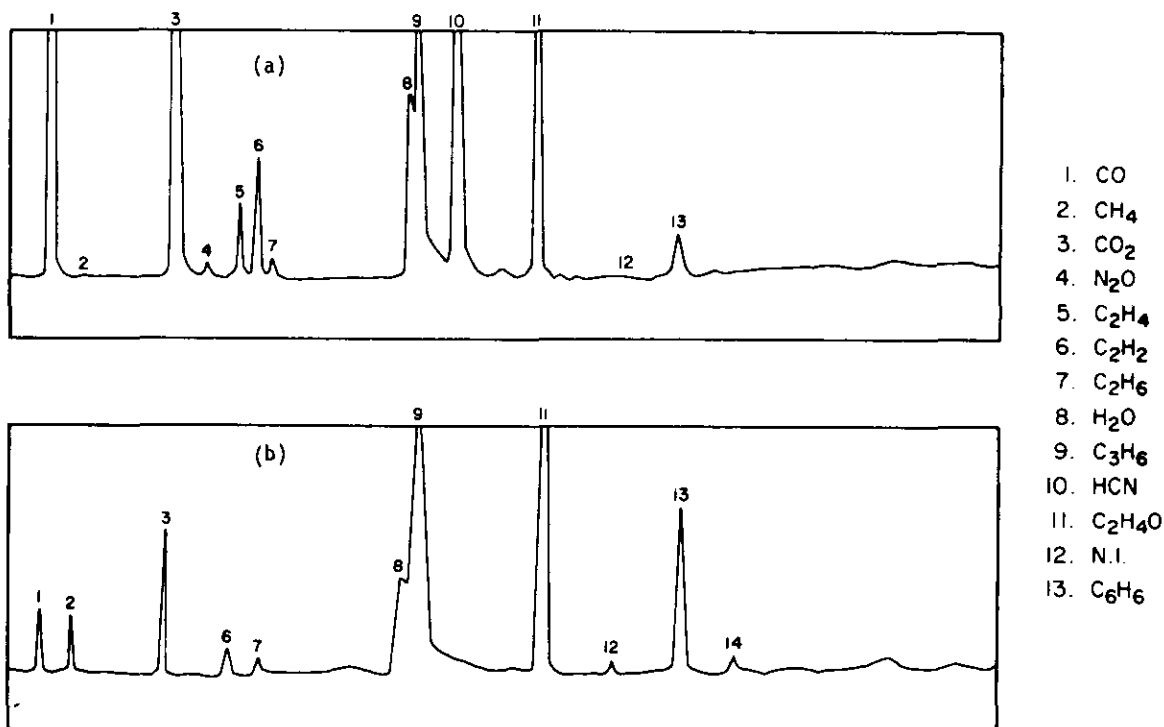9. C_3H_6
10. HCN
11. C_2H_4O
12. N.I.
13. C_6H_6

FIGURE 12. Comparison of degradation products from combustion and pyrolysis of flexible urethane foam: (a) combustion of flexible urethane foam; (b) pyrolysis of flexible urethane foam.

The incorporation of halogen atoms into a polymeric composition, either as an additive or by chemical reaction, decreases the flammability of the material when exposed to an ignition source.

A combination of antimony oxide and halogen is more efficient, as a fire retardant, in many materials, than either of the individual materials alone at the same total concentration. In other words, the combination of antimony and halogen displays an efficient synergism as a fire-retardant system.

The addition of some phosphorus compounds of specific structures retard the burning of many plastics (62).

A combination of phosphorus and halogen compounds or phosphorus and nitrogen compounds may also exhibit considerable synergism as a fire-retardant system.

The most efficient combination of fire retardants varies considerably, depending upon the chemical structure of the material to which it is applied.

Einhorn (63,64) has presented several extensive reviews covering the chemistry of fire-resistant materials, suppression, and the methodology of fire retardance of polymeric materials.

To date, the major concern of those engaged in the development of fire retardant materials has been the reduction of the ignition tendency and flame propagation. It is becoming well recognized that further studies are necessary to determine the physiological and toxicological consequences resulting from exposure to the thermal degradation products of materials containing fire retardants.

Table 18 illustrates the differences which were observed (19) in the nature and concentration of degradation products during the pyrolysis and combustion of a urethane polymer system with and without the incorporation of a reactive fire retardant. Similar differences have been observed during the thermal degradation of natural and synthetic materials.

Conclusions

Improved laboratory techniques have been developed and are being used to provide fundamental information relating to the thermal decomposition of materials. A basic experimental protocol has been established and is being used in the laboratory to evaluate the relative toxicity of materials. The index of potential hazard thus developed is being compared to those parameters encountered by humans during actual fire exposure.

The methodology reported here will be applied to large-scale fire simulation tests to determine the effects of composite materials in situations designed to model ignition and fire growth. Hopefully the results of this research will provide information which can then be utilized in the development and promulgation of more realistic hazard standards for evaluating materials to be used in the "built-environment." An effective premarket screening procedure coupled with a realistic protocol considering both risk analysis and cost effectiveness and coupled with an engineering systems approach encompassing early warning detection equipment and a fire suppression

Table 18. Identification of pyrolysis and combustion products and relative abundance.

Peak	Pyrolysis[a]		Combustion[a]		Identification
	Foam N-4[b]	Foam R-4[c]	Foam N-4[b]	Foam R-4[c]	
1	L	L	L	L	CO (+air)
2	S	L	—	Trace	Methane
3	L	L	L	L	CO₂
4	—	—	S	S	Nitrous oxide
5	L	L	S	L	Ethylene
6	Trace	Trace	S	L	Acetylene
7	L	L	Trace	S	Ethane
8	—	—	S	Trace	NI[d]
9	L	L	L	L	Propylene
10	L	L	L	L	Water
11	—	—	L	L	Propyne
12	S	S	—	—	Methanol
13	—	—	Trace	L	HCN or triazine
14	L	S	—	—	Acetaldehyde
15	—	—	S	S	NI
16	—	—	S	S	NI
17	—	S	S	S	NI
18	—	—	S	S	1-Butene-3-yne
19	Trace	L	—	—	Ethanol
20	—	—	S	L	Chloroethane
21	—	—	Trace	S	NI
22	L	L	L	L	Freon 11
23	L	S	S	—	Propanal
24	—	—	—	S	NI
25	—	—	S	—	NI
26	—	—	S	L	Propene nitrile
27	—	—	S	—	NI
28	—	—	L	L	Benzene
29	L	S	—	—	Dihydropyran
30	S	—	S	S	Toluene

[a] L = large; S = small.
[b] Non fire-retarded rigid urethane foam.
[c] Fire-retarded rigid urethane foam.
[d] Not identified.

system will reduce the toll in lives and property extracted on society as a consequence of unwanted and uncontrolled fires.

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